Hyperinsulinism. This page devoted to glucose concludes with the thesis that hyperinsulinism is a link between smoking and atherosclerosis. All the references do not relate to smoking except (141) Szanto, who has published two letters in the British Medical Journal (16 April 1966 and 15 July 1967). The work cited in the second letter has not appeared in a regular article.

Smoking and Atherosclerosis

SIR,—In a previous letter on this subject (16 April 1966, p. 984) I suggested that smoking and dietary sugar affect the arteries in a similar way. Further work with the help of heavy-smoker volunteers indicates that the atherogenic effect of smoking might lie in its ability to induce hyperinsulinism. This conclusion is based on the following experiment.

Twelve male and seven female volunteers were chosen with the only criterion in their selection that they habitually smoked 20 cigarettes, or more, per day. Their ages ranged from 25 to 57 years. After explaining the purpose of the trial, the subjects were asked to fast overnight and abstain from smoking until a fasting blood sample was taken. Each subject then smoked two cigarettes in succession while talking to each other or reading magazines. A second specimen of blood was then withdrawn. After that, each subject was given 100 g. glucose in water, and further specimens of blood were collected at set intervals for blood glucose and serum insulin estimations. According to the original plan, subjects volunteered to abstain from smoking for 14 days after the first part of the experiment, but only three males and two females were able to do so. The above test was then repeated on these subjects. Glucose levels were estimated by the method of Folin and Wu, and serum insulin by immunoassay.

In the accompanying Table are shown the blood glucose and serum insulin levels in the fasting state, before and after smoking two cigarettes, and during the glucose tolerance test. The values found during the period of heavy smoking and after the cessation of smoking for 14 days may also be compared.

If excessive insulin response can be defined as insulin levels rising above 100 microunits per ml. serum during an oral glucose tolerance test[1] then subject No. 4 may not be considered to have hyperinsulinaemia. However, the marked drop in insulin response after she stopped smoking for 14 days indicates a relative hyperinsulinism during the period of heavy smoking. The view that hyperinsulinaemia is atherogenic is well documented.[2-5] The suggestion that it is the factor responsible for the liability of heavy smokers to develop atherosclerosis is an expansion of this theory.—I am, etc.,

Comparison of Blood Glucose and Serum Insulin Levels

Subject and Sex	Stage of Trial	Glucose (mg./100 ml. Blood)					Insulin (μu./ml. Serum)				
		Fasting		30 min.	60 min.	120 min.	Fasting		30 min.	60 min.	120 min.
		Before Smoking	After 2 Cigarettes				Before Smoking	After 2 Cigarettes			
1 M	30 cigarettes day ..	92	100	151	112	90	87	96	150	156	132
	Stopped 14 days ..	73	78	136	105	70	29	32	54	67	47
2 M	30–40 cigarettes day	85	90	127	103	84	52	53	112	100	41
	Stopped 14 days ..	66	83	112	100	80	35	43	63	57	35
3 F	25–30 cigarettes day	95	95	134	119	85	99	95	145	155	94
	Stopped 14 days ..	54	81	126	102	50	86	84	112	110	41
4 F	20–30 cigarettes day	90	95	145	120	87	33	30	95	84	40
	Stopped 14 days ..	73	108	141	115	60	8	11	26	22	13
5 M	50 cigarettes day ..	78	84	135	100	69	53	50	149	124	63
	Stopped 14 days ..	69	87	137	97	90	30	34	66	72	64

STEPHEN SZANTO.

Department of Nutrition,
Queen Elizabeth College,
London W.8.

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Smoking and Atherosclerosis

SIR,—Your leader (26 March, p. 755) is a fair comment on the present uncertainty with regard to the effects of smoking on the coronary arteries. "On balance," you state, "the evidence is in favour of smoking being a cause, but it is still incomplete, and it would be greatly strengthened if the physiological and biochemical effects of smoking could be shown to contribute to the development of some parts of the disease process."

In a paper published earlier this year[1] it was shown how heavy smokers depend, in certain cases, on their nicotine consumption to maintain their blood sugar level within normal limits. When these people attempted to break with the habit they developed hypoglycaemic symptoms, and to counteract this they ate sweets in a quantity that was surprising even to themselves.

Recently it has been reported by several workers that refined carbohydrates increase the tendency of the blood platelets to stick to the arterial walls. If nicotine is interchangeable with the refined carbohydrates in maintaining the blood sugar on comfortable levels, is it not plausible that it can also cause an increased platelet stickiness in a similar way? To give this hypothesis a biochemical backing, it is known that nicotine exerts an antidiuretic effect due to its action on the hypothalamus. In a present, as yet unpublished, series of tests it was found that the excessive ingestion of glucose or sucrose by carbohydrate-deprived subjects may inhibit for more than four hours the diuresis that is normally expected following the drinking of a litre of water.—I am, etc.,

STEPHEN SZANTO.

Hertford County Hospital,
Hertford.

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